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CITATION:

KAWAMURA, JUNICHIRO. Filling Abnormalities of Intracranial Arteries (so-called vasospasm) : Angiographical Analyses of 63 Cases with Intracranial Aneurysms. 日本外科学会誌 1966, 35(4): 706-718

ISSUE DATE:

1966-07-01

URL:

<http://hdl.handle.net/2433/207318>

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Filling Abnormalities of Intracranial Arteries (so-called vasospasm) : Angiographical Analyses of 63 Cases with Intracranial Aneurysms

by

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Received for Publication May 10, 1966.

INTRODUCTION

Reversible segmental or diffuse narrowing of a vessel near an intracranial aneurysm which has recently ruptured is a common finding on angiograms. Such a narrowing of the artery has been referred to vasospasm, but the correctness of its use has been a great controversy⁸⁾¹⁰⁾¹⁵⁾¹⁸⁾²³⁾. FLETCHER, TAVERAS and POOL⁸⁾ defined that spasm is a narrowing of the lumen of the vessel beyond that which they consider a normal anatomical variant. In the case of the supraclinoid portion of the carotid siphon, narrowing is not considered significant unless its diameter is less than two-thirds the size of the intracavernous portion, as supraclinoid portion of the carotid is always two-thirds or more that of the intracavernous portion. ECKER and RIEMENSCHNEIDER⁵⁾ indicated that the artery is in spasm, when the cerebral artery shows a smaller caliber at angiography with the same technic on different occasions. These descriptions may be acceptable clinically in the arteriographic diagnosis of spasm, but there are several pitfalls in making diagnosis of spasm with these criteria, as JOHNSON, POTTER and REID suggested¹⁰⁾.

Despite of these elaborate descriptions made by other investigators, the authors have attempted to show a new aspect of consideration of the abnormal visualization of the cerebral arteries in an effort to analyze its effects on clinical course of the patients with ruptured cerebral aneurysms.

MATERIALS

The data available in this paper were obtained from the clinical cases of cerebral aneurysms, verified angiographically at Department of Neurosurgery, Kyoto University Medical School, by the end of August, 1964. Sixty-three cases were studied. Aneurysms of the internal carotid artery were found in 22 patients, another 28 cases showed those of the anterior cerebral (anterior communicating) artery, and other 13 patients were revealed to have aneurysms of the middle cerebral artery. The patients with ruptured cerebral aneurysms, verified only by autopsy, were excluded.

METHOD OF ANALYSIS

FLETCHER, TAVERAS and POOL⁶⁾ reported that in their series of 100 cases of intracranial aneurysm there were 38 patients showing spasm following aneurysmal rupture, of which 29 (76%) were studied within three weeks of bleeding. On the basis of this report, the patients studied in this series were divided into two groups: one group includes the patients admitted within 3 weeks after last aneurysmal rupture, and the other those admitted more than 3 weeks later. In most of the cases angiographic examination was performed within two to 3 days after admission. In each of these groups conscious and unconscious (all the patients with impaired consciousness on admission were included) groups were subdivided. Prognostic evaluation of available cases depends on their physical condition on their disposition: improved (or survived) or dead. The abnormal fillings of the cerebral artery were classified as follows (see Fig. 1):

1. CAROTID TYPE (Type C): Abnormal narrowing or poor filling, or both, of C_1 and C_2 . This is often associated with types A-I and/or A-II.

2. ANTERIOR TYPE (Type A): All abnormal fillings of the anterior cerebral artery are included and are divided into 4 groups.

1) Type A-I: Abnormal narrowing or poor filling of A_1 .

2) Type A-II: Poor filling or non-filling of the anterior cerebral artery, or of both A_2 and A_3 .

3) Type A-III: Non-filling of the anterior cerebral artery on one side, and good visualization of both anterior cerebral arteries on the other side.

4) Type A-IV: Non-filling of the anterior cerebral artery, because the aneurysm is too big.

3. ANTEROMESIAL TYPE (Type AM): Poor filling or abnormal narrowing, or both, of the anterior and middle cerebral arteries, especially that of both A_1 and M_1 .

4. SPECIFIC TYPE (Type S): Localized narrowing, or tapering, of the artery just proximal or distal to the aneurysm.

ANALYSES OF THE CASES

1. Aneurysm of the anterior communicating artery.

Twenty-eight cases were studied (Table 1).

1) Within 3 weeks after aneurysmal rupture, 20 patients were hospitalized and examined. Among these, 13 cases were revealed to have impaired consciousness on admission, of which 10 cases, including 8 fatal cases, showed an abnormal filling of cerebral vessels on angiograms. Seven cases without impaired consciousness included 3 cases with filling abnormalities, of which 2 cases were fatal.

2) More than 3 weeks after the onset of symptoms 8 patients were admitted and examined. Two cases showed impaired consciousness and another 6 cases were alert on admission. In the latter group, only 1 case showed filling abnormality, which was dead.

Of 28 cases with ruptured aneurysm of the anterior communicating (anterior cerebral) artery 14 cases (50%) showed abnormal fillings of intracranial arteries, of which

I. CAROTID TYPE

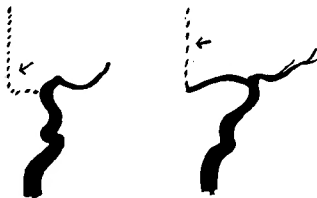


2. ANTERIOR TYPE

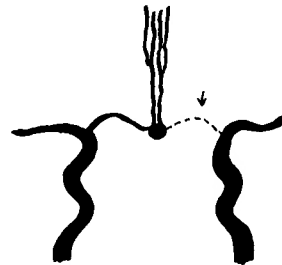
1) TYPE A-I



2) TYPE A-II



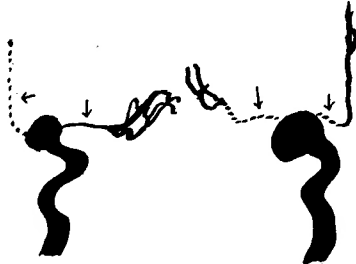
3) TYPE A-III



4) TYPE A-IV



3. ANTEROMESIAL TYPE



4. SPECIFIC TYPE



Fig. 1. Illustration of classified filling abnormalities of the intracranial arteries.

Table 1. Aneurysms of anterior cerebral (anterior communicating) artery

Last Onset to Admission	Number of Cases	Impaired Consciousness		Abnormal Filling on CAG	
		yes	no		
0~3 wks.	20 (14)	yes	13(9)	10(8)	13(10)
		no	7(5)	3(2)	
more than 3 wks.	8 (1)	yes	2(0)	0(0)	1 (1)
		no	6(1)	1(1)	
Total	28 (15)				14(11)

() number of death

11 cases (78.5% in 14 cases) were fatal (in 3 cases the operation was thought to be a major factor of their death), while other 14 cases without such filling abnormalities included only 4 fatal cases (28.5%). In 3 cases carotid type abnormality was found, in which type A-II abnormality was associated with in 2 and both types A-II and AM in 1 case. Abnormal narrowing or poor filling of A_1 (Type A-I) was found in 3 cases, and type A-II, not accompanied by other abnormalities, in 4 cases. One-sided filling of both anterior cerebral arteries (Type A-III) was shown in 3 cases, all of which were revealed to have brain edema, resulting in death. A specific-type filling was noticed in 1 case in which a big fusiform aneurysm at A_3 was found. This case was fatal, because of intraventricular hemorrhage.

The most prominent feature in this group was that 8 out of the 10 cases with types A-II and A-III were fatal. All of the 8 cases with impaired consciousness on admission within 3 weeks after aneurysmal rupture which were revealed to have one or more types of filling abnormalities could not survive.

2. Aneurysm of the middle cerebral artery.

Thirteen cases were studied (Table 2).

1) Six patients were hospitalized and examined within 3 weeks after the onset of symptoms, of which 3 cases showed impaired consciousness on admission. Abnormal filling on arteriograms was noted in 2 cases: one in unconscious group and the other in

Table 2. Aneurysms of middle cerebral artery

Last Onset to Admission	Number of Cases	Impaired Consciousness		Abnormal Filling on CAG	
		yes	no		
0~3 wks.	6 (0)	yes	3(0)	1(0)	2(0)
		no	3(0)	1(0)	
more than 3 wks.	7 (1)	yes	1(0)	1(0)	3(0)
		no	6(1)	2(0)	
Total	13(1)				5(0)

() number of death

conscious group. There was no fatal case.

2) More than 3 weeks after aneurysmal rupture 7 cases were examined. Impairment of consciousness on admission was noted only in 1 case which was revealed to have abnormal filling of the cerebral artery. Other 2 conscious patients showed abnormal filling on arteriogram, but there was no death.

Of these 5 cases which showed abnormal fillings of the cerebral vessel type A-I abnormality was found in 1 case which was associated with type C abnormality. Filling abnormality of type C was found in another 2 cases, one of which was accompanied by type S. One-sided filling of bilateral anterior cerebral arteries was found in one case, and in the last one a large aneurysm at the bifurcation caused non-filling of the anterior cerebral artery.

3. Aneurysm of the internal carotid artery.

Twenty-two cases were studied (Table 3).

1) Fourteen patients were hospitalized within 3 weeks after the onset of symptoms. Four cases had impaired consciousness on admission, all of which showed abnormal filling on angiograms. Two cases were fatal, one of which expired 4 days after carotid ligation. Other 10 cases without impaired consciousness, including one fatal case, did not show filling abnormalities except in 1 case.

2) More than 3 weeks after aneurysmal rupture 8 cases were studied. All of them were alert on admission. Two cases showed abnormal visualization of cerebral arteries. There was no fatal case.

Of the 22 cases with aneurysm of internal carotid artery (intracranial portion) 7 cases showed filling abnormalities of the cerebral artery. In 2 cases type C abnormality was found, which were associated with either type AM or type A-III. Another case which showed type A-III abnormality was accompanied by type S abnormality. Type A-IV abnormality was found in 1 case and type AM without other filling defects in 3 cases. Two out of 4 cases with type AM abnormality were fatal.

Table 3. Aneurysms of internal carotid artery

Last Onset to Admission	Number of Cases	Impaired Consciousness		Abnormal Filling on CAG	
		yes			
0~3 wks.	14(3)	yes	4 (2)	4(2)	5(2)
		no	10(1)	1(0)	
more than 3 wks.	8 (0)	yes	0 (0)	0(0)	2(0)
		no	8 (0)	2(0)	
Total	22(3)				7 (2)

() number of death

RESULTS

1. CAROTID TYPE (Table 4).

Eight patients showed this type of abnormality. Two cases died from intraventricular hemorrhage, resulted from ruptured aneurysm of the anterior communicating artery (Case S. N. became comatose after admission and angiogram done thereafter revealed this type of abnormality). Other 6 cases had an uneventful hospital course. All these patients were operated on.

This type of filling abnormality is frequently accompanied by other types of abnormality, such as types A, AM and S. This may or may not be called vasospasm.

Table 4. 1. Type C (Carotid Type)

	Case	Sex	Age	Impaired consciousness	B. p. †	CSFP †	Prognosis	Findings	Other Abnormal Filling on CAG
*A	S.N.	M	38	No	124/60	110	Dead	I.V.H.	Type A-II
N	S.A.	M	30	No	130/90	300	Improved	I.C.H.	Type A-II, AM
T	S.F.	M	37	Yes	134/68	190	Dead	I.V.H.	Type A-II
*M	Y.K.	F	32	No	134/70	90	Improved		Type S
I	I.I.	M	31	Yes	135/85	280	Improved		
D	K.W.	M	28	Yes	146/110	300	Improved		Type A-I
*I	I.S.	M	46	Yes	120/78	170	Improved		Type AM
N	F.S.	F	29	Yes	110/64	240	Improved		Type A-III

*Sites of aneurysms.

† On admission.

I.V.H. : Intraventricular Hemorrhage

I.C.H. : Intracerebral Hemorrhage

2. ANTERIOR TYPE (Tables 5).

1) Type A-I : Abnormal narrowing or poor filling of A_1 was found in 4 cases, 3 of which had aneurysms of the anterior communicating artery. All of the patients in this group showed impaired consciousness on admission, but only one case with a ruptured aneurysm of the anterior communicating artery died postoperatively. This patient was noticed to have brain edema at operation.

2) Type A-II : Poor or non-filling of both A_2 and A_3 , or of entire anterior cerebral artery, was found in 7 cases, all of which had aneurysms of the anterior communicating artery. Four patients with impaired consciousness on admission took a fatal course (only case T. H. was not operated on). Three other cases involved in this type were alert on admission, but one patient died from intraventricular hemorrhage and another patient developed uncontrollable bleeding during operation, resulting in death. Only 1 non-fatal case had intracerebral hematoma which was evacuated with improvement.

This finding suggests that type A-II abnormality may prospect the worst prognosis of the patients, especially when they are unconscious. This type of abnormal filling is most likely to be produced by increased intracranial pressure rather than a high blood pressure.

3) Type A-III : One-sided filling of both anterior cerebral arteries at angiography may not be specific in case of ruptured aneurysm. Three patients with aneurysm of the

Table 5. 2. Type A (Anterior Type)

1) Type A-I

	Side*	Case	Sex	Age	Impaired Consciousness	B.p.	CSFP	Prognosis	Findings	Other Abnormal Filling on CAG
A	I	H.N.	F	58	Yes	135/78	235	Dead	Brain Edema	
N	I	S.I.	F	43	Yes	120/62	240	Improved		
T	B	I.H.	M	30	Yes	120/70	250	Improved		
M	I	K.W.	M	28	Yes	146/110	300	Improved		Type C

2) Type A-II

A	I	S.N.	M	38	No	124/60	110	Dead	I.V.H.	Type C
	B	S.A.	M	30	No	130/90	300	Improved	I.C.H.	Type C
	B	S.F.	M	37	Yes	134/68	190	Dead	I.V.H.	Type C
N	C	T.A.	M	44	Yes	160/90	450 ↑	Dead	I.V.H.	
	C	T.H.	M	29	Yes	190/130	550	Dead		
T	C	O.N.	M	64	Yes	138/100	235	Dead		
	I	H.Y.	F	54	No	126/80	175	Dead	profuse bleed- ing on Op.	

* Side : Ipsilateral, or
contralateral, or bilateral to the aneurysm.

3) Type A-III

	Side	Case	Sex	Age	Impaired consciousness	B.p.	CSFP	Prognosis	Findings	Abnormal Fillings on CAG
A	C	Y.F.	M	27	Yes	120/90	550	Dead	Brain Edema	
N	C	M.K.	F	47	Yes	165/128	165	Dead	Brain Edema	
T	C	M.A.	F	16	No	134/70	180	Dead	Brain Edema	
M	C	H.K.	F	57	No	160/110	100	Improved	Arteriosclerosis	
I	I	S.Y.	M	66	No	100/72	165	Improved		Type S
N	I	F.S.	F	29	Yes	110/64	240	Improved		Type C

4) Type A-IV

M	I	S.M.	M	52	No	132/88	80	Improved		
I	I	M.M.	F	48	No	164/80	165	Improved		

anterior communicating artery showed this type of abnormality, all of which were revealed to have edematous brain on operation, and took a fatal course. Two of these 3 cases had impaired consciousness on admission, and another case was alert on admission, but took a down-hill course postoperatively. Of 3 other cases involved in this group one had aneurysm of the middle cerebral artery and the rest of the cases had that of internal carotid artery. They were all up and about.

This type of abnormal visualization of the artery may be produced by hypoplasia of anterior cerebral stem.

4) Type A-IV : There were only 2 cases of non-filling of the anterior cerebral artery, which was apparently caused by an unusually big aneurysm of the internal carotid or of the middle cerebral arteries. These 2 cases had an uneventful postoperative course.

3. ANTEROMESIAL TYPE (Table 6).

Poor filling or abnormal narrowing of both A_1 and M_1 was seen in 4 cases with aneurysm of the internal carotid artery and in 1 case with that of the anterior communicating artery. Two of these patients were dead. This type of filling abnormality may be produced by so-called vasospasm, but localized compression of the arteries by blood clots or other mechanical factors may be considered.

4. SPECIFIC TYPE (Table 6).

This type was seen in 3 cases. The patient with a large fusiform aneurysm of the anterior cerebral artery died from intraventricular hemorrhage. Another case had the aneurysm of the middle cerebral artery and the other that of the internal carotid artery. There was no fatal case. This type of filling abnormality may be attributed to "vasospasm", but is more likely to be produced by specific lamellation of the flowing opaque medium before or after filling the aneurysmal dilatation.

Table 6. 3. Type AM (Anteromesial Type)

	Case	Sex	Age	Impaired consciousness	B.p.	CSFP	Prognosis	Findings	Other Abnormal Fillings on CAG
A	S.A.	M	30	No	130/90	300	Improved	I.C.H.	Types C & A-II
I	A.K.	M	65	No	114/60	45	Improved	Brain Edema Arteriosclerosis	Type C
N	K.T.	M	38	Yes	130/90	200	Dead		
	K.F.	F	62	Yes	110/70	?	Dead		
T	I.S.	M	46	Yes	120/78	170	Improved		

4. Type S (Specific Type)

A	T.Y.	M	30	Yes	150/100	500 ↑	Dead	I.V.H	Type C Type A-III
M	Y.K.	F	32	No	134/70	90	Improved		
I	S.Y.	M	66	No	100/72	165	Improved		

I. C.H. : Intracerebral Hemorrhage

I. V.H. : Intraventricular Hemorrhage

DISCUSSION

Many experimental studies so far³⁾⁴⁾⁷⁾¹¹⁾ have demonstrated that in experimental animals mechanical and electrical stimulation of the intracranial arteries produces localized or diffuse narrowing of the vessels, which has been termed "vasospasm". On direct observation of intracranial arteries of human subjects occurrence of cerebral arterial spasm has been also reported¹⁷⁾. At angiography CHUSID et al.²⁾ first reported the appearance of transient hemiplegia as an immediate complication of diodrast cerebral angiography and concluded that the condition is presumed to be due to cerebral vascular arterial spasm.

In fact, most of the neurosurgeons who are familiar with angiographic procedure may have experienced immediate complications of angiography, such as nausea, vomiting, headache, loss of consciousness, generalized seizure, transient hemiparesis and the like, especially when radiographic projection was performed after repeated trials of puncture of the artery, or with perivascular leakage of contrast media. To prevent these complications POPPEN²²⁾ suggested that arteriography should not be carried out under local anesthesia unless the superior cervical sympathetic ganglion and carotid sinus have been thoroughly anesthetized with procaine before the contrast medium is introduced. However, it has not proved yet whether or not they are actually provoked by cerebral arterial spasm.

Practically, the commonest occurrence of cerebral vasospasm (maybe, more precisely called filling abnormalities of cerebral arteries) has been known in cases of recently ruptured cerebral aneurysms. Mechanisms of so-called vasospasm have been considered to be muscular contraction of the artery produced by its mechanical stretch^{3) 4) 7) 10) 11) 12) 13) 19) 20) 21) 25) 23) 27)}, or by chemical stimulation^{9) 24)}. The most popular definition of cerebral vasospasm seems to be referred to that made by FLETCHER, TAVERAS and POOL⁶⁾: By spasm they mean a narrowing of the lumen of the vessel beyond that which they considered a normal anatomical variant. The narrowing involved the intracranial arteries almost exclusively, the cervical portion being rarely affected. In the case of the supraclinoid portion of the carotid siphon, narrowing is not considered spasm unless a local segment only is involved. In the case of the supraclinoid portion of the carotid siphon, narrowing is not considered significant unless its diameter is less than two-thirds the size of the intracavernous portion, as measured on the films. In 100 normal carotid angiograms, they stated, the diameter of the supraclinoid portion of the carotid was always two-thirds or more that of the intracavernous portion⁶⁾. ECKER and RIEMENSCHNEIDER⁵⁾ considered that the artery is in spasm when the cerebral artery showed a smaller caliber at angiography with the same technic on different occasions. This definition is likely to be more reasonable than that made by FLETCHER, TAVERAS, and POOL⁶⁾, but when we read an angiogram on which an abnormal visualization or narrowing of the artery is demonstrated, we have to wait for another angiogram which may show normally filled artery, to make a correct diagnosis of cerebral vasospasm. WOOD²⁸⁾ graded the degree of spasm as severe, moderate, or mild. Severe spasm usually implies an extreme degree of arterial narrowing, often uneven or segmental, with point of almost total occlusion, giving the vessels a beaded appearance in the angiogram. The other extreme is a slight but distinct narrowing of vessels, as often is seen along the course of the middle cerebral artery where its branches emerge from the sylvian fissure. The similar definition of vasospasm was made by MEYER¹⁴⁾ who stated that when the degree of narrowing of the arterial lumen exceeds 50% of its normal diameter, it may be called spasm, and when it remains less than 50% of the normal variant, he calls it vasoconstriction.

Despite of these descriptions, it is not clear that the angiographically defined "vasospasm" actually represents the truly contracted artery. JOHNSON, POOTER and REID¹⁰⁾ mentioned three pitfalls in the arteriographic diagnosis of spasm: (1) congenital or arteriosclerotic narrowing of the artery; (2) temporary narrowing of the artery produced by stretch of a vessel round an intracerebral clot or a large aneurysm; and (3) the ap-

pearance of narrowing produced by variations in blood flow. Other probable causes which may produce a filling abnormality of the artery similar to "vasospasm" includes localized subintimal hemorrhage, localized thrombosis or embolism, direct compression of the artery by blood clot under arachnoid bands, technical variations of the angiographical procedure, or the like. On reading angiograms which show abnormal filling of intracranial arteries, everyone may have had trouble to decide whether or not it might be classified as "vasospasm". Although POOL²⁰) emphasized the importance of cerebral vasospasm in the diagnosis, surgery and prognosis of ruptured aneurysms, the recognition and grading of "spasm" is based largely on the experience and judgement of the observer.

In this study the filling abnormalities of the intracranial artery are classified into four main types, according as their locations and appearances. In the filling abnormalities described as types C, A-I, A-II, AM and S there may be included so-called cerebral vasospasm. Further collection of materials will add other types of filling abnormalities to this classification. With these criteria 26 out of 63 cases studied showed at least any one kind of these filling abnormalities, 20 of which were examined within 3 weeks after aneurysmal rupture. In the 26 cases with filling abnormalities 13 cases (50%) were fatal, in 4 of which surgical manipulations were thought to be a probable cause of death. Other 37 cases without such filling abnormalities included only 6 fatal cases (16%).

On 4 patients no operation was performed. All of these cases died from ruptured aneurysms of the anterior communicating artery. Only one case in this group showed filling abnormality on angiogram (type A-II). The mortality rate is more than twice in the first group with filling abnormalities (50%, but when the operative deaths are excluded, it reaches 34.6%) than in the latter group (16%).

The most frequent occurrence of these filling abnormalities was represented by carotid type which implies abnormal narrowing or poor filling, or both, of C_1 and C_2 . This type was found in 8 cases, the 2 fatal cases of which had ruptured aneurysms of the anterior communicating artery and were associated with type A-II abnormality. The next commonest type was type A-II, which included 7 cases. All of these cases were revealed to have aneurysms of the anterior communicating artery. The most prominent feature of this type of abnormality consists in the highest mortality rate: 6 out of 7 cases were expired (one operative death is included). The next interesting cases had type A-III abnormality which implies bilateral visualization of the anterior cerebral artery on one side and non-filling of the artery on the other. This type was found in 6 cases, 3 of which had ruptured aneurysms of the anterior communicating artery. All of these 3 cases were noticed to have brain edema, resulting in death. Other 3 cases could survive.

These filling abnormalities thus classified may show a new aspect of consideration of so-called cerebral angiospasm and of other abnormal visualization of the artery which have long been drawing attention of neurosurgeons. Practically it is very convenient to classify these filling abnormalities at their sites and appearances. However, the classification just mentioned is still inadequate to come to general agreement and should be modified in future.

In this classification type A-III abnormality is most likely to be produced by hypoplasia of the anterior cerebral stem. RIGGS and RUPP²⁶⁾ analyzed 994 specimens of the

complete circle of WILLIS which had been obtained from adults presenting clinical manifestations of neural dysfunction, and found 119 specimens of hypoplasia of proximal stem of one anterior cerebral artery. Type A-IV abnormality is apparently produced by a physical factor. Other types of filling abnormalities may include so-called vasospasm, but other mechanical factors must be considered. For instance, in type A-I abnormality hypoplasia of the anterior cerebral stem should be ruled out, and in type A-II abnormality there may be poor or non-filling of the anterior cerebral artery produced by increased intracranial pressure¹⁾¹⁶⁾. In fact, 4 out of 7 cases which showed this type of abnormality were revealed to have either intraventricular or intracerebral hemorrhage.

It is rather easy to make the angiographical definition of cerebral vasospasm, but is very difficult to find out which filling abnormality really represents a true "vasospasm". The authors think that the larger intracranial arteries have possibility to show spasm, and defines conveniently that a localized abnormal narrowing of the artery is "angiographically" termed vasospasm. We might as well use the term, vasoconstriction, for a physiological vascular contraction evoked by Bayliss effect.

SUMMARY

Sixty-three cases with aneurysms of the internal carotid (22 cases), the anterior communicating (28 cases) and the middle cerebral arteries (13 cases) verified angiographically at Department of Neurosurgery, Kyoto University Medical School, by the end of August, 1964 were studied.

The filling abnormalities of cerebral arteries were classified as follows:

1. Carotid type (Type C): Abnormal narrowing or poor filling, or both, of C_1 and C_2 .
2. Anterior type (Type A):
 - (1) Type A-I: Abnormal narrowing or poor filling of A_1 .
 - (2) Type A-II: Poor filling or non-filling of the anterior cerebral artery, or of both A_2 and A_3 .
 - (3) Type A-III: One-sided visualization of bilateral anterior cerebral arteries.
 - (4) Type A-IV: Non-filling of the anterior cerebral artery, because of a too large aneurysm.
3. Anteromesial type (Type AM): Poor filling or abnormal narrowing, or both of the anterior and middle cerebral arteries, especially that of both A_1 and M_1 .
4. Specific type (Type S): Localized narrowing, or tapering, of the artery just proximal or distal to the aneurysm.

With this classification 26 out of 63 cases showed at least any one kind of these filling abnormalities, 20 of which were examined within 3 weeks after aneurysmal rupture. In the 26 cases with filling abnormalities 13 cases (50%) were fatal, (including 4 probable operative deaths), while other 37 cases without such abnormalities included only 6 fatal cases (16 %). Type C abnormality was found in 8 cases (2 fatal cases), Type A-II in 7 cases (6 fatal cases) and Type A-III in 6 cases (3 fatal cases). The highest mortality rate was shown in Type A-II abnormality and 4 out of 7 cases with this type of filling abnormality were revealed to have either intraventricular or intracerebral hemor-

rhage.

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和文抄録

脳動脈瘤における脳動脈の造影異常 (いわゆる血管攣縮) に就いて

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いわゆる脳血管攣縮の定義, 発生機序等については, 従来種々論議がある。著者は, 脳血写学に於ける脳血管の造影異常という見地から, 昭和40年8月末までに, 京大脳神経外科で, 脳血管写により脳動脈瘤を証明しえた63例について, この問題を検討した。

内訳は, 内頸脳動脈瘤(頭蓋内)22例, 前大脳(前交通)動脈瘤28例, 及び中大脳動脈瘤13例である。

脳血管造影の異常を下記の如く分類した。

1. Carotid Type (Type C): C_1 及び C_2 の異常狭窄又は造影不良, 或いは, その両者を示すもの。

2. Anterior Type (Type A): 下記の四群に分つ。

1) Type A-I: A_1 の狭窄又は造影不良を示すもの。

2) Type A-II: 全前大脳動脈又は A_2 及び A_3 の狭窄又は造影不良を示すもの。

3) Type A-III: 一側からは前大脳動脈は造影されず, 他側から両側前大脳動脈が造影されるもの。

4) Type A-IV: 動脈瘤が, 異常に大きいため, 前大脳動脈瘤が, 前大脳動脈まで, 造影されないもの。

3. Anteromesial Type (Type AM): 殊に A_1 及び M_1 の狭窄又は造影不良を示すもの。

4. Specific Type (Type S): 動脈瘤の直前又は直後で, 局所性狭窄を認めるもの。

以上の分類によると, 63例中26例が何らかの型の造影異常を示し, その中20例は, 動脈瘤破裂の最終発作から3週以内に入院したものであり, 死亡例は13例(50%)であつた。又上記の造影異常を示さなかつた残り37例中, 死亡例は, わずか6例(16%)であつた。Type C を示したものの8例(死亡2例), Type A-I を示したものの4例(死亡1例), Type A-II 7例(死亡6例), Type A-III 6例(死亡2例), Type AM 5例(死亡2例), Type S 3例(死亡1例)であつた。特に高い死亡率を示した型は, Type A-II で, 1例の手術死を含め, 7例中6例が死亡, 殆んど例に, 脳室内出血又は, 脳内血腫を認めた。

上記の脳血管の造影異常の分類は, まだ不十分ではあるが, ある程度予後判定の基準になりうると考える。

本論文の要旨は, 第23回日本脳神経外科学会で発表した。